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Vol. XXV] JULY 1958 [No. 1

PAIN IN THE FOOT

by

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FEET have to be kept in a sound condition. People in South India walk bare-footed in a very large majority of cases.

The common causes of foot pain are :-

1. Abnormalities of the metatarsal arch and flat foot.
2. Hallux valgus complex and hallux varus complex.
3. Hallux rigidus.
4. Hammer toe.
5. Severe corns.
6. Ingrowing toe-nails and ungual exostosis.
7. Pain (*a*) after fractures of the metatarsals, phalanges and tarsal bones (*b*) after fractures of the leg (*c*) the scarring due to loss of skin over ankle joint (*d*) disorders of circulation.
8. Pain and corns due to unreduced dislocations of the metatarso phalangeal and inter-phalangeal joints.
9. Acute conditions from fungus infection, lymphangitis in the bursa of untreated tallips, Rheumatic fever, infective arthritis, infections.
10. Chronic inflammatory conditions due to fibrositis, Madura foot Gonorrhoea, Diabetes, Gout and Tuberculosis.
11. Thrombo-angitis obliterans and other circulatory disorders as frost bite, early stages of diabetic gangrene.
12. Calcaneal spur.
13. Calcification of tendo achilis and inflammation of the bursa in front of the insertion of the tendon.

14. Neuritis due to diabetes, Leprosy and injuries in nerves.

15. Foreign bodies inplantation dermoids, fiproma, fibrosarcoma, Tuberculosis of tendon sheath, Sarcoma of bones.

16. Tumours and aneurysms in popliteal space pressing on nerves and referred pain from tumours of spinal cord.

Pain in the inner border of the foot, in the position of the tarsal scaphoid bone in a child of 7 to 10 is due to osteochondritis of the navicular, tuberculosis of the bone has to be excluded in adolescents and in other periods of life, flat foot is the common causes of pain.

Heel pain. 1. Pain on the under surface of the heel is felt especially during the weight-bearing ; sometime the pain may be agonising ; there may be localised pain at one spot. Patients are sometimes young about 20 and generally above 40. Radiogram may show a calcaneal spur. The origin of the spur is due to a mild periostitis the result of an infective process like rheumatism. Fibrositis due to diabetes mellitis and gonococcal infections are common. These spurs may disappear spontaneously. After operations they may reform. Calcaneal spurs may be bilateral but pain is complained of only in one side of the foot. Local warmth, relief from pressure by a felt having a hollow in the centre at the situation of the spur are useful. Fibrositis cases respond to local warmth and cortisone by mouth.

2. **Pain at the back of the heel.** This is a common complaint at all age periods. The Secondary epiphysis of the os-calcis appears at about the age of ten and joins at the age of sixteen. An ache especially after walking or games is complained of. Radiograph shows osteo-chondritis of the secondary epiphysis. Sloping of the heel of the shoe to relax the tendo-achilis is required with general treatment. Tuberculosis of the os-calcis is common in childhood. There may be recurrent attacks of pain. Old fractures of os-calcis often cause pain in the sub-astragaloid joint and round about the os-calcis due to the formation of the new bone. Theseare obstinate types of cases and will require arthrodesis of the subastragaloid joint.

The posterior surface of os-calcis is divided into three zones—an upper which is smooth and separated from the tendoachilles by a bursa ; a middle rough and vertically ridged for the insertion of tendo-achilis ; and a lower which is continuos with the tubercles on the plantar aspect and supports the flat of the heel. There is often bursitis of this bursa ; radiograms show ossification of the insertion of tendo-achilis to a varying degree from small streaks to large plaques varying in length and breadth. These changes are often seen in elderly people. If the radiogram shows atheromatous vessels, nothing should be done to the foot not even manipulations. The pain in the foot is due to deficient circulation and thrombo-angitis obliterans, atherosclerosis should be always remembered and excluded. Rest in equinus position, local warmth and strapping give relief of pain. Acupuncture is sometimes of value. Removal of the bursa or ossified bone is very rarely indicated.

Flat Foot. It may be stated that a large majority of the population suffer from symptoms referrable to foot which may be described as incompetent to carry on its functions. The functions of the normal foot are weight bearing during standing and locomotion, the provision of a spring to reduce jars and assist propulsion during movement and action as a pivot to assist the turning of the body.

A foot may be anatomically a sound foot but may become functionally

incompetent. Flat-foot may be either congenital or acquired. Congenital flat foot may be the result of anamolies in the development of bones or deficient development of tone of the tibial muscles.

Acquired flat foot is due to :—

1. Toxins of acute illnesses as pneumonia, diphtheria, whooping cough, measles, smallpox and chickenpox.

2. Toxins of chronic sepsis as infected tonsils and infected teeth.
3. Excessive heat as in stokers.

4. Excessive cold as trench feet, thin socks.

5. General muscular fatigue due to over - marching, standing, high-heeled shoes and cramped shoes.

6. Malnutrition as in rickets with poor muscle tone.

7. Rabid growth ; muscular development and tone do not keep pace with the growth of bone.

8. Abnormal lines of stress due to bony deformity as external rotation of the lower limb due to hip condition, genu valgum, genu varum, and genu recurvatum, injuries to the bones of the leg and the ankle joint where proper alignment has not been restored.

9. Trauma to the bones of the foot as from fractures or disease of the bones.

10. Poliomyelitis of the tibialis anticus and posticus, spastic pralysis and paralytic causes.

11. Congenital deficiency of the invertors and calf muscles (calcaneo-valgus)

12. Two muscle conditions in the leg which mechanically produce flat foot are shortening of the extensor longus digitorum from injuries as scalds and deep burns and shortening of the tendo-achilis. A strain on a tight tendo-achilis tends to pull the foot into an everted valgus position.

Congenital type of flat foot. This is associated with a cavus foot (i. e.) too deep an arch. Subluxation of the inner part of the head of the talus is a fixed deformity and severe in cases of congenital flat foot.

Signs and symptoms. Earliest signs are an uncomfortable hot feeling and if they are wearing boots they complain about the unsuitability of the boots giving rise to discomfort. The foot feels stiff towards the end of the day's work. There is a tendency to walk with the feet everted. There is no inclination to rise on the toes. They feel better after a day's rest. There is relief by inversion of the foot. Localised pain and tenderness under the tubercle of the navicular is one of the early signs. This is due chiefly to the stretching of the plantar calcaneo navicular ligament. Pain and tenderness is felt below the internal mallcolus, below the talo-calcaneal joint and on the dorsum of the tarsus ; they may complain of pain along the tibialis posticus due to tenosynovitis. The gait of the patient becomes alerted. The raising of the heel is avoided during walking to prevent stresses on the tarso-metatarsal ligaments. Patient carefully lifts the ball and heel of the foot at the same time to spare the ligament. They may walk by rotating the feet and legs outward. Synovitis of the knees and back ache with pain in the buttocks and back of legs are common. They get easily fatigued and complain of cramps in the feet and legs. There may be swelling in front of and below the external malleolus and in front of the tendo-achilis.

Flat foot may be classified into different stages. In the first stage the patient is voluntarily able to correct the deformity. In the second stage the Surgeon is able to correct the deformity without an anaesthetic. If there is acute pain or the patient is examined after a heavy exertion there may be considerable spasm and pain but the arch can be restored after a few days rest or under an anaesthetic. In the third stage the deformity has become fixed and there may not be much pain due to the loss of the arch itself but complications may be present.

Examination of the case. Children should be examined for deformities of the knee as knock-knee and coxa vara. In the school-going adolescent period recent history of infections as tonsilitis, diphtheria and influenza should be enquired into. Rapidly growing children have to be examined for flabby muscles and wrong posture. In the examination of adults in addition to knock knee and coxa vara history of injury to the ankle joint, tarsal and metatarsal bones should be excluded. Focal sepsis and residual infection in the urethra and prostate should be eliminated. The pain of the thrombo-angitis obliterans brings the patients into orthopaedic out-patients. There are many cases where foot prints show the presence of the arch but patients complain of severe pain. This is common in the hardworking agricultural classes and city labourers. It is appropriate to call these feet as inefficient and they become subjected to repeated foot-strain which in turn produces adhesions or arthritic changes.

Complications of flat foot. Corns in various sites are common, a corn under the head of the first metatarsal, a hole in the sole of the shoe in the same region, indicate faulty weightbearing. A corn on the inner side of the great toe indicates that the shoes are too short. Corns under the heads of all the metatarsals represent failure of the intrinsic muscles of the sole to keep the toes flexed and the transverse arch up. Plantar flexion of the matatarso-phalangeal joints is due to the intrinsic muscles i. e. subluxation of the heads of all metatarsals. If the heel balance is faulty i.e. a valgus position of the heel, a corn on the internal malleolus will be seen. Arthritis of the first metatarso-phalangeal joint with stiffness is due to constant weight bearings as this joint was never designed for weight-bearing and hallux valgus with arthritis follows. Subluxation of the inner part of the head of the astragalus follows as soon as the calcaneum tilts into valgus. Spasm of the peroneii, short tendo-achilles and mild arthritis with adhesions of any or all of the foot joints are seen.

Prophylaxis of the flat foot. 1. No patient should get up after acute illness in soft " bedroom slippers " but should wear a firm boot or shoe with laces. When the patient stands up after convalescing from any acute illness, it will be seen that his inner-arch sinks down. If this is neglected an entirely preventable condition becomes a permanent rigid foot. 2. Reduction of the body weight and correct posture of the back. 3. Improving the balance of the foot by postural re-education.

A position of constant external rotation of the hip whether postural or fixed will deflect an undue share of the body weight on to the inner border of the foot and thus disturbs the normal muscular equilibrium very unfavourably. Fixed internal rotation by favouring the anti-gravity muscles as tibialis anterior and posterior are less harmful. The external rotation of the lower limb should be prevented and this requires watchful care.

The common causes of external rotation are congenital dislocation of hip whether unreduced or reduced, coxa vara

due to slipping of the epiphysis, fracture of the neck of the femur or bending by rickets, osteomalacia, cysts and arthritis of all types. Genu valgum. genu varum and recurvatum have the same effect of throwing the foot into valgus. They should be prevented. Deflections of the limb below the knee due to rickets and malunited fractures affect the foot. Forward and backward bowing of leg affect the subastragaloid joint which is extremely sensitive to the least alteration in alignment. Fractures of one or both bones of the leg should be carefully treated to obtain correct alignment of the knee and ankle joints.

Flat foot in children. In children the arch cannot be considered flat until after the age of 24 months. Upto this time a thick pad of fat fills the arch and makes it appear flat. The arches of the foot are present at birth.

If the ankles roll inward so that the inner side of the shoes are worn off, correction must be done. The mother brings the child for weak ankles (i.e.) the child's ankles turn easily and cause him to fall. Sometimes the mother brings the baby for a prominence on the inner side of the foot i.e. this prominence is the head of the astragalus which has rotated inward and downward i.e. the forepart of the foot has rotated outward. Children do not complain of painful arches but leg aching at night and the mother notices less activity of the child in play. Even in double flat feet of severe grade, the child may have no symptoms though in later life severe disability is likely to occur.

Children with weak arches should not go barefooted. Gymnasium shoes should not be worn by one with weak arches.

Treatment. Correct rickets, focal infection, arthritis and obesity. Local measures of correction of a flattened arch consist in keeping the forepart of the foot from rotating outward. In children upto 5 years old, tilt the shoes with a strap of leather $\frac{1}{8}''$ thick, tacked along the inner border of the shoe sole. Stretching exercise in the milder cases is useful. (*a*) Force the foot upward against the leg fifteen or twenty times while the foot is turned inward and the knee is held straight. (*b*) Stand with toes turned slightly inward then sit down on the heels which must be kept flat on the floor. Repeat slowly fifteen to twenty times. Correction under an anaesthetic with application of casts for six weeks is useful in the more marked cases.

Shoes. The babies shoe must be broad with flat flexible sole with slight or no heel and lace front. The toes should lack about $\frac{1}{2}''$ of reaching the tip of the toe of the shoe. Length, width of the toe, depth of the toe, depth of instep, and gripping at heel must always be present if the shoe is to give comfort and mould the foot properly. Wearing high heels, short fashionable shoes at the ages of 10 to 14 causes such foot distress in later life as deformity and corns. If the ankles turn easily, top shoes should be worn and the ankle part braced by lining it with stiffening material. Poor class of children are treated with plaster of paris moulds after correction.

Treatment. The principles of treatment are restoration of muscle balance by attention to general body posture and rest, suitable diet with vitamins, correction of faulty alignments of the limb as genu valgum by calliper or bony faults by ostectomy, exercises to re-educate the inversion of heel and also heel and walking. Tip toe exercises are bad. Local rest to the foot by correct shoe with good toe room, large heel, close fit to instep and laces is required. In cases with acute pain or severe dropping strap to hold the metatarsals together ; felt under a thin stiff insole is useful. If there is an outward slope of the os-calsis

crook the heel of the boot and add a small wedge under the head of the first metatarsal. A sandal or strap shoe with a crooking does not take enough grip of the foot and is not of any use. In children the crooking of shoe gives great comfort. Complications should be treated only after the correction of flat foot. Adhesions are broken down under anaesthetic at several sittings so as not to provoke reaction. Peroneal spasm may need correction under an anaesthetic and the corrected position is maintained by plaster of paris application. The external dropping of the inner side of the foot in congenital and rickety cases is improved by prolonging the inner side of the heel. Sub-astragaloid and mid-tarsal arthrodesis is very useful in the stiff and painful feet which are usually severe and neglected cases. This had to be done in the poor type of Indian agriculturist class when manipulation had failed.

On Shoes. The foundation of a shoe is the insole which is the part of the shoe with which the sole of the foot comes into contact, this should be carefully made and must not be irregular. A cast which is a block of wood of the shape of the foot is obtained : the sole of the last should be regular and smooth. To make the insole, a piece of leather is soaked in water till it is mellow after which it is rolled or hammered out, cut to the desired shape and then hammered to the sole of the last and kept in position by nails. While the insole is in position. the upper with its lining is fitted over the last and sewn to the insole and the wedge of the upper is sewn to the welt. The welt is a strip of leather about an eighth of an inch thick and half an inch to three quarters of an inch broad running round the sole as far as the heel. It forms a backing to which the outer sole can be sewn without taking the stitches, through the upper. The space between the insole and the outer sole is filled with a composition of saw-dust and glue. This sometimes becomes glued and irregular due to heat so that the insole becomes uneven, boat-shaped from side to side as it drops in the centre and causes pain. In front of the heel is the waist, which should not break or sag and allow the cuboid bone to drop, this should be made of leather only. Steel or wooden shank pieces are not good as they are too rigid. Rigidity is not good but the insole should be resistant. Sandals have no resistant force. Rubber and canvas are not resillient like leather and there is no resistant force in such shoes. The heels should be flat with a good supperficial area in contact with the ground. Regarding high heels one and half inches may be allowed ; very pointed heels are bad because of the difficulty of balancing. The heels should not come so far forwards as all the time the person is tending to overbalance backwards. As a large number of persons have short tendo-achilis a moderately high heel is an advantage. The soles should be sewn and not nailed because nails destroy the welt and make further repairs difficult and unsatisfactory. In the shoe the outer part of the foot should strike the insole first and the inner arch is left quite free. The toes should be able to spread and heads of the metatarsals should not drop. Instep grip is absent in court shoes (i.e.) shoes with no fastening across the instep.

(*To be continued*)

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CEREBRAL VASCULAR CATASTROPHY

DIAGNOSIS AND TREATMENT

by

Dr. R. SUBRAMANIAM, M.D., M.R.C.P.,
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Introduction

With the advent of chemotherapy and anti-biotics infectious diseases have been brought considerably under control and mortality due to these diseases have been reduced considerably in living memory. Yet, the degenerative disorders are not under control. Though we know some factors, still we are not in possession of full details with regard to the control of degenerative disorders like coronary thrombosis, cerebral vascular disorders and other diseases. They are common as age advances.

For instance, cancer, coronary thrombosis and cerebral vascular catastrophy were not so common as they are at present. This is so because as age advances, the vessels do become brittle, less elastic and less capable of adapting themselves to circumstances and strain. Atheromatous changes occur in the endothelial surface promoting easy formation of thrombosis. The vessels thus become more tortuous so that this action favours thrombus formation. This tortuosity itself is due to the loss of elasticity. It is an uncoiling as it were of a blood vessel which is no longer capable of dilating and accommodating the extra load of blood that is thrown into it by each systole.

When called upon to see a person who has suddenly lost power ever one half of the body, it means that a vascular catastrophy has occurred. It is only in a vascular catastrophy that sudden onset of paralysis can occur. In no other condition can the onset of paralysis be so abrupt as in the case of a blood vessel going out of action. So a sudden onset of paralysis must always make one think of the possibility of a vascular lesion. The lesion may be haemorrhage, thrombosis or embolism. Of the three phenomena, again, the clinical manifestations are likely to be very abrupt in onset with an embolic process. This is comparable to the closing of a tap. When the tap is closed, water-supply is cut out quickly—so this manifestation hardly takes seconds, for the active blood flow is stopped abruptly. Generally these emboli have their origin from the heart. In rare cases of auricular septal defects, paradoxical embolism can occur, i.e. the emboli can start from the systemic veins and be carried up and be shunted from right to left and result in a systemic arterial block. This is comparatively a rare incidence of embolic manifestation. This is more of academic interest than of practical importance. In contrast to embolism, in cases of haemorrhage the onset of paralysis takes some minutes. In spite of the leak, some amount of blood is still being carried by the blood vessel. The lack of blood supply will only be gradually felt in a case of cerebral arterial thrombosis, it is not likely to be so abrupt as in an embolic manifestation but even then it is fairly quick. It takes time for the thrombus formation. There is always a certain amount of patency, so that the paralysis is not generally complete and also it takes some hours for it to develop.

When called upon to treat a case, etiology of the paralysis must always be borne in mind. If it is a case of haemorrhage nothing much can be done. Progressively the paralysis will get worse. To start with, patients generally complain of headache and in a short time they become unconscious with progressive loss of consciousness. As time advances, coma deepens. Cerebrovascular haemorrhage has aptly been compared with that of a river in floods. As soon as a vessel ruptures, blood gushes out of it. It peels off the soft brain matter around it, and when the blood reaches the next blood vessel it peels off the brain that is supporting the vessel. Now the second vessel is left without a support. Owing to the leak of blood, there is a tendency for the intracranial pressure to rise. If the systemic pressure rises, the rate of escape from the area of rupture increases. Besides the second vessel, which is now without a support, has to sustain this high pressure. Generally it does not stand the strain and ruptures and result in a fresh haemorrhage. Now the two vessels are ruptured—in other words two rivers are in floods. Practically the major portion of the brain is destroyed and life ceases due to failure of the respiratory centre. In most cases of haemorrhage, this course of events takes place. Rarely the haemorrhage may get localised and the man might live. This is a rare event and it is not of common occurrence. If a cerebral vascular catastrophy has occurred, and the patient has survived that by a few days for all practical purposes, cerebral haemorrhage can be ruled out. In most cases of cerebral haemorrhage, death occurs within 24–48 hours. Sudden death is not the feature in cerebral haemorrhage. It generally takes 24 hours or longer. Rarely it may end fatally in as short a time as one hour and 45 minutes.

So when one is called upon to see a case of extensive paralysis. it is always worthwhile treating the case as cerebral thrombosis. This attitude is assumed so that the treatment may be adopted assuming that the patient is likely to live. The first thing that has got to be done is to do a lumbar puncture and get the C. S. F. If the lumbar puncture shows clear fluid that itself will eliminate cerebral haemorrhage. There is a false notion among some practitioners that lumbar puncture might provoke further haemorrhage. Such a thing does not occur. On the other hand letting out a fluid even in a case of haemorrhage results in lowering of the intracranial pressure. This in turn lowers the systemic pressure. If the systemic pressure is also lowered it will favour arrest of haemorrhage. On the other hand, in cases of cerebral thrombosis, there is likely to be a certain amount of oedema. This oedema causes increased intracranial pressure and headache. Both will be relieved by lumbar puncture. So lumbar puncture can safely be done with advantage in both conditions. In all cases of cerebral haemorrhage, particularly, where no history is available it is worth while taking an X-ray of the skull to eliminate intracranial neoplasms and fractures of the skull bones.

Though in most cases an X-ray of the skull will be of very little value, yet the patient has got to be protected against the dangers that inevitably accompany mental confusion. Careful and skilful nursing is essential for the first few days. Side railings should be used for the cots as long as the patient is unconscious. Basal pneumonia is likely to occur, in these cases and the same can be prevented by Penicillin injections of 3 lakh units daily, for the first few days, and by frequently changing the position of the patient. Particularly during the first few hours it is advisable to keep the patient on the side with no pillows. This is done to prevent the tongue from falling backwards and choking. Another very essential thing in unconscious patients is to prevent them from getting dehydrated. This is best done by passing

a Ryle's tube and administering nourishing liquid feeds by drip. I. V. drip must be avoided as it might precipitate pulmonary oedema. In the course of 24 hours 4·5 pints of fluid should be administered.

Bladder must be cared for from the very beginning. Even in hemiplegic patients, retention of urine can occur. The mechanism of this retention is not well explained. If retention of urine occurs causing distension of bladder, an indwelling catheter with tidal drainage is the basis. Repeated catheterisation will result in sepsis. If on the other hand spontaneous voiding of urine occurs, then change of the sheets be made as often as necessary. If the respiration is irregular or shallow, if there is cyanosis, oxygen 95 parts and carbondioxide 5 parts as a mixture can be given for inhalation. The idea in giving carbondioxide is to stimulate the respiratory centre.

Also caffein et sod. benzoas 1½ gr. or strychnine 1/30 gr. can be given I. M. every 4 hours.

As soon as the patient regains consciousness; steps should be taken to encourage him to use the paralysed limbs. From the very beginning, passive movements must be given in all the joints on the paralysed side. This prevents arthritis and the pain of disuse is bound to occur if neglected at this stage. The shoulder joint is especially susceptible to develop pain. If in spite of passive movements pain develops, then dry heat of diathermy or massage can be tried. Massage done in the proper way on the hemiplegic side, not only relieves pain but also helps in the recovery of the paralysed limb. This is a fairly simple procedure and any hospital should have a Masseur on its staff. In a number of my cases, they were considerably relieved of pain by massage.

Treatment of associated conditions like diabetes mellitus or heart disease or obvious infections, should also be corrected in cases of cerebral thrombosis. At any rate the heart will need supportive line of treatment, and without treatment for the cardiac condition chances of the patient being able to move about are very poor.

Once the patient is able to use the paralysed limbs, re-education of the paralysed limbs in the correct way should be started. To start with, an over-correction is done so that ultimately it will be normal. For instance the hemipligic patient has got a tendency to circumduct his leg. He should be taught to actively flex his leg and then move forward. If he actively flexes his leg, then it will be an exaggeration of the normal flexion that occurs in the knee. Though such a step may be awkward to start with it will finally result in a normal style of walking. So also the defects in the speech should be corrected. One of the best ways to rehabilitate is to send him back to his old job because he knows best what he has got to do and thereby he gets self confidence. But for this, to start with, he should be given only part-time work and gradually the work must be increased by prolonging the hours of work each week. In the early stages of thrombosis there is vascular spasm.

In some cases, the spasm may pass off without the thrombosis setting in. In those cases, the paralysis is transient. If called upon early, the patient is put on 95 per cent oxygen and 5 per cent carbondioxide mixture for inhalatien for 5 to 10 minutes each hour. This may be given whenever the patient is conscious or comatose.

Procaine block of the stellate ganglion daily for a few days, considerably aids clearing of the hemiplegia. There are numerous procedures for doing stellate ganglion block. In my own cases I was

not much impressed by this procedure. Not only is there an element of risk in this procedure but also sudden death may occur. So before adopting this procedure, its seriousness must be explained to the relatives and then only attempted.

When the diagnosis of thrombosis is definitely made, anticoagulants like Dicoumerol or Tromexan may be employed for 6 weeks. The dose of the drug is to be controlled by repeated blood examination for prothrombin time. After the first few days are over, the etiology of the thrombosis is found out and the same is treated. Generally, in the younger age group, it is syphilitic; in the older age group hypertension, arterio-sclerosis and diabetes mellitus may operate. At any age group polycythemia vera or even polycythemia secondary to cardiac malformations might be an important cause. To improve the circulation of the infarcted area, vasodilators are administered. This consists of administering 1 mg. of histamine, 300 mg. of nicotinic acid and 500 mg. of ascorbic acid in half pint of 5 per cent glucose solution or glucose saline. Glucose saline is used for patients with low B. P. or in normal blood pressure cases. In hypertensive cases 5 per cent glucose solution is used. The whole thing is mixed together and administered as drip. It should be allowed to flow in very slowly and for giving this 10 oz. at least 2 hours must be taken. In most cases, the effect is very dramatic. The earlier this is given better the results. In the last eight years, I have used this for over 800 cases with extraordinarily good results. In the early stages, this can even be given twice or thrice daily but due to lack of facilities in most cases I have given only once a day. In many of them, patients were able to walk in the course of a week or ten days. Recovery is particularly well marked in the younger age group, more so when they are of syphilitic origin. Even in cases who have had hemiplegia of some years duration, they were able to recover to a considerable extent. In a small percentage of cases, where the improvement is not very marked, the treatment was repeated about a month later, with considerable improvement. The action of histamine seems to promote vasodilatation of the adjacent vessels, i.e. vessels adjacent to the thrombosed vessel, so that the affected part gets its blood supply from the collateral circulation, resulting in considerable improvement. Unless the hemiplegia is of long standing duration some degree of improvement was observed in all the cases treated by me, either in the hospital or outside. The effect of this administration is generally to cause a fall in the systemic pressure. Sometimes the fall may be as much as 30 mm. of mercury (both systolic and diastolic).

In one of my cases. Mr. S. Ch. he had coronary thrombosis and was under treatment for nearly six months. I was suddenly called upon one day to find that he had developed hemiplegia on the right side. An interesting feature was observed by me in this case. Seeing that cerebral thrombosis had occurred only a few hours ago, I suggested giving this patient histamine by drip along with vitamin C and nicotinic acid. This was given for 12 days in all. With this treatment the patient recovered not only from the hemiplegia but by the time the treatment was complete, he also recovered to a considerable degree from the coronary insufficiency he had. Probably histamine therapy had dilated the vessels in the coronary circulation as well. I have not tried this in my other cases which came subsequently I fear that in an acute attack of coronary thrombosis there is already a fall in the blood pressure and histamine therapy will cause further a fall in the blood pressure. I feel this therapy may be tried cautiously after the first few days are over and the patient has got over the shock.

2 to 3 oz. of whisky may also be given. It also causes vasodilatation and a sense

of well-being in the patient (only this is not available in prohibition areas). Papaverine 0·2 gms. every 4 hours I. M. may also be given with the idea of causing vasodilatation. In a small series of cases, I was not impressed by the effect of Papaverine to the same extent as I was with histamine therapy.

Rehabilitation of the hemiplegic patient. Passive movements may start at the end of 24 hours in thrombosis cases. Where the diagnosis is haemorrhage, three weeks rest is essential. Start the exercises slowly. At no time should the patient get tired. At first every few minutes of exercise must be followed by prolonged rest. The period of exercise is increased gradually. Patients who can use the affected upper limb as also patients who can lift the affected lower limb off the bed can walk. When the paralysed leg can be lifted off the bed, it means that there is sufficient motor power in that leg to support the body for standing as well as for walking. But the hemiplegic patient who has been in bed for some time may hesitate to walk. He is likely to feel nervous and lack confidence in himself. In such cases the patient must be reassured and also supported.

One of the most convenient ways that I have found for aiding these patients to walk is to push a wheel chair with a patient. The patient in a wheel chair not only acts as a dead-weight but also controls the speed of the chair. Besides this, a bamboo or a stick can be tied at waist height and the patient may be asked to grasp the same and walk about. When once he walks without much difficulty he can be given a walking stick with a blunt end and be asked to move about.

Prevention of Deformities. Footdrop must be prevented by posterior leg splint. Sand-bag is put on the out side of the leg to prevent outward rotation of the paralysed limb. A small pillow in the axilla of the paralysed side will prevent adduction at the shoulder.

Active Rehabilitation :—This will help considerably by a braided U-shaded cord tied to the foot-end of the cot or to a cross bar fixed over the patient's cot. With the help of this braided cord the patient can sit up, lean forwards and backwards and he can repeat these movements several times. He can also be given exercises in which weights are lifted over a pulley. The advantage of the pulley is that the exercises can be graduated. In place of the wheel chair, two kitchen chairs can be placed, one on either side of the patient and by slowly moving one chair after the other, the patient will have a constant barricade on either side of him. At the same time he will have a support. For this purpose the chairs must be light with a straight back but also strong enough to bear the weight of the patient. There are now available series of articles which can be used by partially disabled persons. Such articles should be used by these patients till such time as they are able to use both hands. The most urgent need of the country is to have a large number of rehabilitation centres all over the State.

THE PROBLEM OF VARICOSE VEINS AND GRAVITATIONAL ULCERS

by

Dr. K. A. KALYANAM, F.R.C S., Madurai.

Choice of the Subject :—

1. " The treatment of varicose veins in this country is not much above that of the African Witch Doctor "—These words were used by a famous surgeon in 1949 when delivering the Hunterian oration in the RCS of England. If this was the position in the United Kingdom in 1949, we can well imagine the position in our country.

2. The problem of varicose veins not only concerns the individual but is one of national importance. It is gradually becoming recognised as a troublesome weed, causing much misery to the sufferer and loss of man hours to the industries. The cost to the nation in dressings, drugs and in loss of labour though impossible to estimate, must be considerable.

We cannot yet offer the patient with varicose veins completely satisfactory treatment but we can say that all sufferers can be benefitted ; that the majority of the complifications can be prevented, and a high percentage of those who consult us can be cured.

The condition though not so common as in the Western countries is very frequently met with both in private and hospital practice.

Recent advances in the understanding of the causation of varicose ulcers and indeed the varix itself have modified the treatment to a great extent.

Historical : Nearly 2500 years ago Hippocrates, the legendary father of Medicine was on the whole against the incision of superficial varices. He directed that the varix itsef should be punctured in as many places as possible. This trauma at multiple points may have damaged the intima sufficiently to cause thrombosis. Again sepsis might have played a part in the production of this clotting.

The famous surgeon, writer, philosopher and administrator. Sir Benjamin Collins, Bvodie described the test in 1846 which was destined to receive the name of Trendelenburgh half a century later. Friedrich Trendelenburgh described the ligation of the Saphenous vein in the thigh in 1900 or so and published a result of 22% recurrence rate. George Perthes (1869–1927) studied in Bonn under Trendelenburgh and was well in advance of his time in the investigation of the patency of deep veins.

The history of injection treatment of varicose veins dates back to 1853. Sclerosants have had a chequered career, being discarded towards the end of last century, reintroduced 30 years ago and now after prolonged trail and error once again being relegated to far less important position than they enjoyed in the past. The use of sclerosants in association with operative treatment stood the test for any years but once again this treatment is rapidly becoming of historical interest only. History repeats itself throughout the centuries but it is suggested that the present era is one which holds out a far more optimistic future for the sufferer.

Present day methods are the summation of the varied therapies of the past. The " flush tie " of high resection of the internal saphenous vein was envisaged by Paul of Aegina 1200 years ago, made more definite by Moore of Melbourne during the last century and finally perfected by Homans some thirty odd years, ago. Stripping operations have figured in varied techniques over since the days of Hippocrates. It is only in recent times however that these methods have been made safe. As mentioned before Sclerosants have come, gone, returned to favour, and in our present time once again on their way out.

Anatomy : Four main groups :

1. The deep veins of the leg such as femoral popliteal and tibial.

2. The supperficial veins which empty into the femoral vein at the saphenous opening-the long saphenous system.

3. The superficial veins which empty into the poplitial vein-the short saphenous system.

4. The deep and superficial veins which empty direct into the internal iliac vein. These drain the buttock and posterior part of the thigh.

The superficial and deep groups are connected by communicating veins or perforators. The significance of these communicating veins has been increasingly realised in recent years. In health the blood flows from superficial to the deep system.

Although the anatomy of the superficial system of the lower extremities is most inconstant and variable, their communicating veins are far more constant and variations in position are not nearly so usual. Cockett has shown that there are three constant perforating veins behind the posterior margin of the tibia in the lower third of the leg : lower middle and upper and one at the lower third of the lateral aspect not communicating directly with the saphenous vein. These are of importance in the cuasation and treatment of ulcers which he calls the ' ankle blow out sydrome .'

Venous Valves : The entire saphenous system of veins is plentifully supplied with valves, but the position is far from being constant and there is no regularity about the general disposition : a constant valve is one in the femoral vein just above the point at which the internal saphenous vein joins it. The communicating veins are provided with valves which cause the blood to be directed from superficial to the deep veins. There are valves in the deep veins also.

Physiology : The factors which are responsible for the blood in the veins against gravity are :

1. Muscle contraction : the venous heart or the peripheral heart
2. Capillary pressure,
3. Muscle tone and involuntary contractions
4. Compression from the pulsations from the adjacent arteries.

Incidence and Aetiology : It is well known that Negroes, Indians, Mexicans and Chinees suffer from this condition far less frequently than do the white races. It is most probably due to the fact that we do not spend long hours standing as do to the Nordic race : 10 to 15% of the people in Western countries are said to suffer from some degree varicose veins. Another point of interest is the relative frequency of the disease among men and women. More than 70% of the sufferers are women. Pregnancy, puberty and menopause with associated endoctrine disturbances have something to do with this finding.

Aetiology : The causation of varices has puzzled the minds of investigators throughout the ages. The search for their origin has been clouded by the fact that there is no one single cause. Varicose veins may be found in almost every part of the body. The frequency of their appearance in the legs may make us forget this fact. It is obvious that they may occur in the absence of the mechanical factor.

Recently the matter has been further clarified by the excellent work of Prof. King of Melbourne University. He approaches the whole subject from a new and most interesting angle. He feels that there are two quite distinct stages in the production of varices. He speaks of the :

1. Primary

2. Secondary aetiological factors, and feels that investigators have concentrated too much on the secondary or late aetiological causation and too little on the early stage of the disease. In other words it is no use studying the long established varix showing the typical signs of thrombosis, classification, valvular atrophy, elongation and tortuosity. He shows that long before these changes occur in the veins, there are definite histological alterations which are constant in the very early stage of the varicosis.

King feels very strongly that early stage of varicose veins is a haemodynamic problem and not hydrostatic ; and changes are probably due to a chemical, probably a hormone or hormones of Oestrogen group. He produces evidence to support his theory the details of which are found in his article 'The Genesis of Varicose veins'. Summarising King states that 'in the early stages there is a disturbance of the vascular relationships in an area resulting in an increased arterio-venous blood flow. This is associated with relaxation of the vessel walls not only of the arterio - venous communicating vessels but in the veins themselves. This gives the greater blood flow in the veins with the centripetal pulse, the oxygenated blood, the dilatation just distal to the valves, the reversibility if the chemical conditions alter.

It accounts for the localisation of the condition with the normal vessels to be seen proximal to the affected area.

Taking all these findings into consideration it is clear that the vascular changes are not due to simple mechanical back pressure, but to some stimulus affecting the local conditions of the vessel wall and the blood flow. The immediate stimulus appears to be one of a chemical nature and there is evidence that this chemical substance may well be a hormone or hormones of the Oestrogen group the changes are reversible at this stage and so prevention is in the realms of possibility.

The Secondary Setiological factors are well known. They are :

1. The erect stance of the human as opposed to that of the quadruped

2. The heriditary factor

3. Pregnancy

4. Changes resulting from inflammation of the veins and their valves.

There is no doubt one of the important aetiological factors in the causation of varicose is the retrograde hydrostatic pressure due to the prolonged effect of gravity which has not been adequately opposed by the gravity of the leg muscles. For this reason policeman, waiters, labourers and nurses are most frequent sufferers. No example of the varices occurring in any four legged animal be traced, although it has been reported that a donkey who had died recently at

the London Zoo had suffered from this condition. Adequate confirmation of this point was however lacking.

In pregnancy the varices develop before the effect of the pelvic tumour comes into play and pregnant women suffering from severe varices are less likely to abort than those who are free from varices which emphasises the importance of the endocrine factor.

Pathology : Established varices may present all or the majority of the following conditions :

1. Elongation,
2. Tortuosity,
3. Impairment or absence of elasticity
4. Localised dilatation,
5. Variations in the thickness of the vein wall
6. Valvular atrophy,
7. Localised patches of thrombosis,
8. Areas of clacification.

The majority of cases affect the long saphenous system of veins about 90%.

Investigations :— Many complicated tests have been evolved for testing of the condition of both the deep and superficial venous systems and for the determination of state of communicating veins. These have been evolved to determine.

1. The competence of the valves of the communicating veins,

2. The competence of the valves of the internal or external saphenous system,

3. The patency of the deep venous system. But all the authorities are agreed now that these tests are not of much value and regardless of the results of Trendelenburgh test, treatment of varicose internal saphenous vein should be complete elimination of the vessel.

The normal colour of the foot, without Oedema and without pain on walking (acçording to Homan and Cockett) indicates satisfactory deep circulation of the leg.

As a confirmatory simple test the elastic bandage test can be done. A firm one way stretch bandage is applied from the toes to the knee which obliterates the superficial veins and the patient asked to walk for 10 minutes. If there is no pain the deep veins are patent. It is extremely rare to find deep veins obliterated without a previous history of pain and oedema.

Treatment

Preventives :—At the present time no prophylactic or preventive treatment is known. Treatment by means of adminstration of oestrogens or its antagonistic hormones have not been effective. All patients presenting family history of varices should be regarded as potential sufferers and should be advised to be careful in selection of occupation.

Curative :—Over thousands of years the elusive varix has been a most stubborn foe, and it is only in recent times the subject has become sufficiently specialised to offer our patients not only relief but in the majority of cases that which is commensurate with cure. Even so we must always remember that the disease is of a progressive & degenerative nature and that whatever form of treatment we give, all our patients require periodic supervision. Changing fashions in the treatment of varicose veins have been as frequent over the past fifty years as the changing fashion in ladys' dresses. The pendulum of fashion has swung a full length on many occasions from excision on the one hand sclerosing obliteration on the other.

The Conservative treatment: of rest, elevation and some form of external support is indicated in cases where surgical treatment is not necessary or contraindicated.

Treatment with sclerosant injections :—The controversy regarding this form of treatment has been with us for several years.

Fratkin and Jackes (1951) state that sclerosants are dangerous materials and that the danger of thrombosis to the individual is sufficiently great without introducing a substance known to produce intimal and consequent vascular damage. The use of sclerosants is dangerous and the effect not lasting-and that complete cessation of their use should be practised by all interested in varicose veins." Boyd and Robertson state that there is great danger to the deep venous system and they proved this by special venographic experiments. It is contended however that the right sclerosant, put into the right place in the right (small) amount can still be a valuable adjunct in the fight against the varix.

Indications :—Sir Heneage Ogilvie stated, " I am afraid time has come to consider seriously whether the injection treatment of veins has any place in legitimate therapy. For those who would daily on the slippery slopes of cosmetic surgery it has its attractions; for it is a cosmetic procedure and no more. The injection of a prominent vein will cause that vein to disappear. This is no cure of the disease and this cosmetic problem does not exist in our country. The feeder vein of varicose ulcer and those which have missed avulsion are other indications for injection treatment.

Operative :—Now that the deficiencies of sclerosants are more fully recognised surgical treatment of varices has become the most important method. The one point of common agreement is that operation of high resection of internal saphenous vein is an essential antecedent or part of any of the varied methods of surgical attack which are in common use.

The ' top tie ' or ' flush tie ' of Ameicans consists of careful high resection of internal or long saphenous vein after ligating all tributaries at this point. This is usually followed by complete stripping of the varcies which gives the most satisfactory results.

As regards the choice of the patients or indication for treatment : the athletes or soldiers veins must be removed. The problem of cosmetic veins as I have already mentioned, does not exist in our country. Pregnancy veins require conservative treatment.

The large tortuous veins without symptoms (i.e. without oedema), skin irritation or disability have to be dealt with on their merits. The patient's occupation (i.e. involving long periods of standing, risk of trauma, infection etc.) Proximity to medical facilities, the question of joining the police or armed services should be considerations for treatment. The oedematous, pigmented and ulcerated legs-which are complications-are definite indications for treatment.

Gravitational Varicose, or Venous Ulcers. Three groups :

1. Varicose,
2. Thrombotic,

and 3. Ischaemic.

A varicose ulcer is caused by the incompetence of the valves of the superficial venous system-including the ankle perforators (Cockett) in which there has developed a retrograde circulation sufficient to cause oedema and necrosis.

The Thrombotic or post phlebitic ulcer is a sequel of throbophelebitis affecting the ileo-femmoral, popliteal and deep

leg veins causing destruction of the valves. Both these can be called venous ulcers.

The ischaemic ulcer is caused by arterial deficiency-about which we are not concerned here.

The secondary factors which are present in majority of leg ulcers are

1. Infection (Pyogenic or fungoid),
2. Obesity,
3. Trauma,
4. Lymphoedema,
5. Arterio-venous communication
6. General diseases like Diabetes, Syphilis or anaemia.

The venous ulcers occur only in the lower two thirds of the medial surface and lower one third of the lateral surface of the leg. Any ulcer above these area is not due to gravitational causes.

It is well known that great majority of patients at all ages with varicose veins had no leg ulcers and the great majority with ulcers have no varicose veins. To explain this the recent work of Cockett of St. Thomas Hospital is of great value.

Based on a study of 234 cases of ulcers, he found that in ulcers of venous origin, only 52% had an incompetent great or small saphenous system-two thirds of which had incompetent ankle perforators. 21% had neither incompetent saphenous system or definite history of thrombosis.

He has also proved by careful venographic studies that in the erect position the essential venous drainage of the ulcerbearing area is into the deep veins mainly but not entirely via the three large perforating veins on the inner side and one large perforator on the outer side of the leg. The Saphenous has little place in the venous drainage though by their incompetent valves and varicosity there is a certain amount of added-stasis indirectly. This explains why even radical treatment of varices of saphenous system sometimes or most often does not cure the ulcers.

The thesis that venous ulcers and induration of the lower leg are usually due to destruction of valves in ankle perforating veins is put forward by Cockett and supported by clinical, venographic, anatomical and experimental evidence with or without incompetent saphenous or deep venous system. Based on this thesis an operation of extrafascial ligation of these incompetent perforators and excision of the ulcer if present is described by him, Non-recognition of the importance of these ankle perforators is the cause of recurrence and he has reported very many cases of recurrent chronic ulcers after treatment of varices cured by ligation of these ankle perforators. He calls the oedema, pigmentation and ulceration as ‘ the ankle blowout syndrome ’. For those who do not wish for operative treatment and for those above the age of 50 years and for those who can afford the time and money, the routine conservative treatment is very effective. This is the modified Bisgarrd method of applying the elastic bandage. The most important points are :

1. The education of the patient in the details of applying the elastic bandage.

2. The encouragement of movement and exercise of the leg and calf muscles. The patients should not be put off work or limited in their activities in any way.

3. The avoidance of all irritating applications to the ulcer site-especially the modern antibiotics-Zinc cream and olive oil are the only two applicants allowed.

The Method :-Initial rest and elevation to reduce oedema. The ulcer margins

are massaged with olive oil and zinc cream applied to the ulcer and a piece of dry gauze to cover the ulcer and surrounding skin. A felt pad is applied cut to cover the ulcer, the surrounding skin and extending upwards to give pressure to the region of the ankle perforating veins. Crepe bandage is applied to keep the pad in place and a heavy elastic webbing bandage is applied from toes to the upper portion of the leg. This should be done every day before getting out of bed.

Although the treatment is of immense value it is obviously not suitable for our patient in the Tropics.

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* "IRON METABOLISM, THE EFFECT OF THE MUCOSAL BLOCK AND THE BODY IRON STORES AND THE ASSIMILATION OF IRON FOR THE TREATMENT OF IRON-DEFICIENCY ANEMIA".

by

Mr. P. E. WARNEFORD,

Messrs Benger's Labs. Ltd., England.

THE points which are to be high-lighted in this article are taken from three papers published on this subject and its treatment by the intramuscular preparation known as IMFERON.

You will no doubt be amazed to read that since Imferon was introduced in August 1954 there have been 85 papers written and published on this subject. You will appreciate that we cannot go through this mass of written word. These papers have been written and published in all parts of the western world. Whilst some deal in very glowing terms with the preparation 'IMFERON', others are quite naturally more critical. Yet in every case they admit that in its treatment Iron-Deficiency Anaemia, IMFERON is unique and alone in the treatment of these conditions.

The first of these papers deals with "IRON METABOLISM" and was written and produced by Phillip Sturgeon, M. D. of the Department of Hematology Research, Los Angeles. Let me read you a paragraph from this paper which is headed 'Iron Assimilation':

> "The passage of iron through the intestinal epithelial cell is not a matter of simple diffusion. Within physiologic limits and in most therapeutic conditions, ferrous iron enters the cell and, after oxidation to the ferric state, reacts with the protein apoferritin to form ferritin in which state the iron is retained within the mucosal cell. Then it is released from ferritin, reduced and in the ferrous state enters the circulation. Initially some iron may pass directly into the circulation, but very quickly a "block" develops and additional iron does not gain immediate access to the circulation but may be retained within the mucosal cells as ferritin".

It will be seen, therefore, that the assimilation of iron either from the food we eat or the iron tablets given orally to correct anemia depends entirely on the state of the hemoglobin content of the blood as to whether this iron is released from mucosal cells for the re-generation of red blood cells. Tests have proved conclusively that in the treatment of anemia with patients who have 50 to 60 per cent hemoglobin (on the Haldane scale) oral iron will regenerate the hemoglobin up to a figure of 85 to 95 per cent. But beyond that figure the ability of the body to utilise further supplies of iron by this route is greatly retarded. It will be appreciated that once anemia has shown itself by the lowering of the hemoglobin, the body iron stores and the serum iron are nil. It would be realised therefore that iron given orally will regenerate the hemoglobin up to a point, but will not and cannot, except by months of treatment, permit the re-stocking of the

* Summary of a lecture delivered at the Madras City Branch on 27—5—'57

stored iron which the body retains or should hold against the blood loss by various courses. An entirely different picture is, however, presented with parenteral iron. Benger's Labs. were the first people in the field with an intravenous iron. But we realised that even by this method there were certain limitations and the fear of reactions. The Benger's research team found the perfect method of introducing iron into the human body by the intramuscular route. This preparation is, as we now know, called IMFERON. It is injected into the upper and outer quadrant of the buttock deeply into the muscle and from there it is immediately transported into the blood stream to be made available for the synthesis of hemoglobin.

Let us look for a moment to what F. G. J. Hayhoe, M. D., Lecturer in Medicine, Cambridge University and Honorary Consultant Physician of the United Cambridge Hospitals says:

> " When iron balance is impaired by excessive iron loss through haemorrhage, growth, pregnancy and lactation or other cause, or rarely by inadequate iron intake, the iron reserves of ferritin an haemosiderin in the liver, spleen, bone marrow, and the other sites of reticulo-endothelial cell activity are drawn upon for haemoglobin synthesis. It is not until these reserves, which amount to between 600 and 1,500 mg. of iron in the adult, have been exhausted that the serum iron falls and the intestinal mucosal receptors avidly take up fresh supplies of iron from the diet ".

> " Oral iron is capable of reversing the sequential process to a stage where tissue iron is restored and both the blood picture and serum iron level return to normal, but at that stage the mucosal iron receptors are no longer unsaturated and little further iron can be absorbed from the intestine to reconstitute the iron reserves ".

> ' The effective and relatively non-toxic intravenous iron preparations have recently been supplemented by an equally satisfactory intramuscular preparation, and parenteral treatment offers no difficulty in patients who are refractory to oral iron, or in whom it is important to re-establish iron reserve without delay ".

You will see therefore that by treating iron-deficiency anemia by the i.m. route the whole system of assimilation of iron from the gut the consequent blockage caused by the raising of the hemoglobin in the mucosal cells is by-passed. You have here an effective and efficient method for the regeneration of hemoglobin and at the same time restoring the body iron stores.

Now, for a moment let us look at the cause and effect of anemia. A.R. Stevens M.D. of Seattle, U.S.A., produced a very striking diagram of iron deficiency in childhood, and below this diagram he gives the following quotation:

> " A new born child has 250 ml. of circulating blood of which a 150 ml. are red blood cells. The same child at one year old has the circulating blood of 750 ml. and providing anemia has not set in, the red blood cells total 300 ml.

If, however, because the child has been deprived of any sort of iron during its first year of life, the red blood cells remain at 150 and you have therefore a picture of an anemic child.

We all know that the iron content of milk is notoriously low. Human milk is even lower than cow's milk. It is estimated that only 0·25 ml. of iron are available daily if the child is fed entirely

by its mother. The picture with cow's milk is slightly different and even then the need for additional iron is still there. It has been proved beyond any doubt whatsoever that, for a child to grow naturally without becoming anemic, it must receive during its first year of life 150 mg. of iron of 0·6 mg. daily.

We have seen that the iron which is taken orally is passed from the small intestine to the portal vein and is then transported by the plasma for the synthesis of hemoglobin. We have also seen that IMFERON injected reaches the blood stream within hours from the time of the injection. The iron is then bound to a protein which is available in the plasma for this purpose. The known red blood cells is comprised of four parts of hame or iron, to one part of globin or protein, and the iron which is bound to the protein syderophylin and transported to the red bone marrow for the synthesis of red blood cells. What iron which is available in the blood stream and is not capable of being bound by the protein syderophylin is held by the liver or spleen and is stored there until, in itself, capable of being bound and utilised for the production of further red blood cells.

We saw that iron when converted from ferric iron to ferrous iron in the duodenam and then passed to the small intestine across the mucosal cells must in itself be reconverted back to ferrous iron before being taken up by the blood stream. It will therefore be seen that ferratin which is a water soluble compound is not clearly definable in the body, but haemosederin which when in the red bone marrow being cystalined is capable of staining and it is from this method only that the condition of the body iron stores is capable being checked.

How does Iron Deficieney Anemia Arise ?

There are three causes :

(1) Excess loss of iron,

(2) Difficulty in absorbing iron,

(3) A combination of these two factors.

Let us first of all examine iron loss.

All cells from the body surface and from the lining of the intestines carry some iron with them. Tests which were conducted over 120 days proved conclusively that the average loss was 0·5 mg. per day. It will be seen therefore that with an average male the body loses irou to the extent of ½ mg. per day but a very difficent picture arises when we examine the female body. Here the menstrual loss can be as much as 1½ or 2 mg. per day during the menses. We have seen that the body is capable of absorbing ½ to 1½ mg. dependant on the food and the condition of the absorbing mechanism in the human body. By this means nature holds the body absorbtion in equilibrium. If the loss, however, is greater than ½ to 1½ mg. per day, then the deficiency must be made up by a route other than the oral method. When we looked at Stevens' paper, we saw the necessity of 150 mg. of iron being made available and deposited in the growing child each year. The iron balance under these circumstances is therefore a very precarious one.

Later on we have proved conclusively that the relationship between available iron and body growth is very close. Let us look for a moment at a normal adult blood picture. The body iron in circulation in the blood is 2·5 Grm, and the stores iron 1·5 Grm., making a total of 4 Grm. of iron in the healthy human body. With iron deficiency estimated at 7·5 or 50% (on the Haldane's scale), the blood picture shows iron to the extent of 1·25 Grm. and stores iron nil. We have therefore a deficiency of 2·75 grms. of iron. This picture can be rectified within weeks by parenteral treatment, but it would take months' oral treatment to even

restore the hemoglobin to a normal level and would not take into account the body iron stores which would permit anemia to return should there be any excessive blood losses through any cause.

Let us now look at the female picture. Tests were carried out by medical women in the U. S. A. and they have proved without any doubt that the actual menstrual loss was much greater than had been realised. They went on to prove that as much as 20·3 mg. of iron was lost each day during the period of menses.

In pregnancy, blood is passed from the mother to the child during the three trimesters. In the first trimester, 0·5 mg. of iron are passed from the mother to the child she is carrying. In the second trimester this increases to 7·5 mg. and in the third and final trimester 312·5 mg. of iron are handed over. It will be seen therefore that in prematurity, the inablity of the mother to hand over to a child born prematurely that child must certainly be anemic. Repeated pregnancies at short intervals can cause great loss of iron to the mother. Have you realised therefore that if a pregnant woman attends a Maternal Clinic during the third and final trimester, should there be any signs of anemia it is physically impossible to raise the hemoglobin level before the third stage of pregnancy. The result must be a further drop in the hemoglobin of the mother and the birth of another anemic child. The child will without question suffer from a retarded growth and chronic inflammatory conditions during its early life.

In general, therefore, it will be seen that an anemic patient has lost iron for many years before the Doctor sees him. The hemoglobin was perfectly normal or perhaps only slightly reduced but during its time the body iron stores were being subjected to a constant drain of the iron.

Iron-deficiency anemia can be normocytic and it is only after some months that it becomes hypochronic and microcytic. In general, anemia has started months before. Iron stores are in all probability greatly depleted or completely empty when the treatment for anemia commences. The 1–5 grms. of iron as a reserve to meet this need has been used and should there be a further loss gross anemia will result.

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